Muscle metabolism and PSE pork[1]

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Abstract

Pale, soft, and exudative (PSE) pork is primarily caused by an accelerated rate of postmortem glycolysis resulting in low muscle pH while carcass temperature remains high, thus causing protein denaturation. Numerous factors influence the rate of postmortem metabolism and may be responsible for the rapid pH decline characteristic of PSE pork during the 1st h postmortem. Release of high levels of Ca^{2+} from the sarcoplasmic reticulum into the sarcoplasm triggers an accelerated rate of postmortem glycolysis and is responsible for the high incidence of PSE observed in halothane pigs. Because total muscle ATPase activity is closely associated with rate of glycolysis, differences in myosin ATPase activity may explain in part the accelerated glycolysis in PSE development. Although important in determining the overall rate of glycolysis, differences in glycolytic enzyme activities have failed to explain discrepancies in postmortem metabolism between PSE and normal muscle. Elevated muscle glycogen content and an extended duration of postmortem glycolysis can also result in PSE-like characteristics, as seen in RN^- pigs. Shifts in muscle fiber type toward higher proportions of glycolytic fibers may help explain the incidence of PSE. Inherent differences in mATPase activity, Ca^{2+} regulation, glycolytic enzyme profiles, and glycogen content between muscle fiber types integrate various mechanisms responsible for the abnormal postmortem glycolysis in PSE muscle. In an effort to delineate potential mechanisms responsible for PSE, models to artificially induce PSE, such as retarding temperature decline and electrical stimulation, provide useful tools. The value and limitations of these models stem from their abilities to alter postmortem temperature and pH declines and to effectively simulate the protein denaturation typical of PSE pork.

Key Words: Muscle Tissue, Glycolysis, Metabolism, Pork, Postmortem Changes, Meat Quality

Introduction

Increased selection pressure for leaner pork products in recent years has resulted in leaner, heavier-muscled pigs. Many of the newly established genetic lines are reminiscent, phenotypically, of those prominent in the late 1960s and early 1970s. Unfortunately, meat quality concerns indicative of that same period remain prominent in today's pork industry. One such condition that has received much attention is pale, soft, and exudative (PSE) pork. Pale, soft, and exudative meat is characterized by a pale lean, soft texture, and a low water-binding capacity. The PSE condition is problematic to many facets of the pork industry and contributes significantly to the great variation observed in pork quality today.

Many data have been collected over the years regarding PSE development. Experiments ranging from live animal studies to highly controlled in vitro studies attempting to model PSE development have been performed. The purpose of this review is to summarize briefly the biochemistry behind the transformation of muscle to meat and highlight factors responsible for the aberrant postmortem metabolism that causes PSE. Additionally, this review attempts to expose gaps in the literature as well as provide an overview of potential models to study PSE.

Transformation of Muscle to Meat

Muscle, unlike most other tissues, contains a unique set of proteins that allows it to contract. Although muscle functions in several different capacities, its primary role is to provide a means of locomotion through carefully orchestrated contraction/relaxation cycles. This whole cycling process is modulated by fluctuations in cytosolic calcium (Ca^{2+}) levels. In contracting muscle, Ca^{2+} is released from the sarcoplasmic reticulum and Ca^{2+} levels within the sarcoplasm reach approximately 10^{-5} *M* concentration, whereas in relaxed muscle the concentration is lowered to about 10^{-7} *M* (Winegard, 1968; Inesi and Malan, 1976; Endo, 1977). At contraction-induced levels, Ca^{2+} "instigates" conformational changes in the thin filaments and allows myosin to bind actin molecules. Subsequently, contraction is generated by a series of events in which ATP binds to the myosin head, breaking the actin-myosin bond, and ATP hydrolysis causes a conformational change in the myosin head, resulting in a "swinging" action and the reattachment of the myosin head to an adjacent actin molecule (Huxley, 1974). In order for muscle to relax, Ca^{2+} is removed from the sarcoplasm, via an ATP-dependent Ca^{2+} pump, and is resequestered in the sarcoplasmic reticulum, thereby removing the ability of Ca^{2+} to induce contraction. If ATP is not available in the muscle fiber, irreversible cross-bridge formation between myosin heads and actin occurs and rigor mortis results in the tissue (Bendall, 1951). Thus, ATP and Ca^{2+} can be considered two major

players in muscle contraction and metabolism. That is, ATP is the source of energy for breaking actomyosin cross-bridges and for maintaining the Ca^{2+} pump of the sarcoplasmic reticulum, whereas Ca^{2+} is a primary regulator of contraction.

One of the key metabolic pathways in the conversion of muscle to meat is glycolysis. In living tissue, glycolysis generates ATP for the aforementioned cellular functions as well as a myriad of other activities. In postmortem muscle, the tissue attempts to maintain homeostasis by preserving cellular ATP concentrations, but due to circulatory failure following exsanguination, muscle lacks the oxygen required for oxidative metabolism. Consequently, muscle glycogen is metabolized via anaerobic glycolysis, thus phosphorylating ADP to replenish ATP. Anaerobic glycolysis is less efficient than aerobic metabolism at generating ATP. Thus, as postmortem metabolism continues, glycogen and ATP levels decline, and lactic acid accumulates, lowering muscle pH (Kastenschmidt et al., 1968). This process results in an overall pH decline to an ultimate pH (pH_u) of about 5.4 to 5.7 at 24 h in pig longissimus muscle (Briskey and Wismer-Pedersen, 1961).

Development of PSE pork is a pH- and temperature-dependent phenomenon (reviewed by Sebranek and Judge, 1990; van Laack and Solomon, 1994). The PSE condition develops in muscle primarily due to an accelerated rate of glycolysis early postmortem while carcass temperatures are still high. The subsequently lower pH at elevated temperatures immediately after slaughter results in greater protein denaturation than in "normal" muscle (Briskey and Wismer-Pedersen, 1961; Charpentier, 1969; Goutefongea, 1971). Such denaturation results in the elevated water loss (Offer, 1991) and altered light scattering characteristic of PSE muscle (Goldspink and McLoughlin, 1964). Additionally, PSE-like characteristics may develop due to a prolonged postmortem glycolysis (Monin and Sellier, 1985).

Regulation of Glycolysis in Postmortem Muscle

Numerous factors have been investigated as possible causes for the abnormal postmortem glycolysis in PSE muscle. Although not completely independent of one another, the potential mechanisms resulting in an aberrant glycolysis may be categorized into four areas: Ca^{2+} regulation, muscle ATPase activity, glycogenolytic enzymes, and substrate (glycogen) regulation. Additionally, muscle fiber type is an underlying factor encompassing the aforementioned points of potential glycolytic regulation.

Ca^{2+} Homeostasis. Cytosolic Ca^{2+} concentration within muscle fibers plays a major role in muscle metabolism and is a vital component contributing to the triggering of rapid postmortem glycolysis and PSE development. Calcium accelerates glycolysis by two mechanisms: increasing activity of Ca^{2+}-activated ATPases and(or) serving as a cofactor in many glycolytic pathway reactions. Pigs with porcine stress syndrome (**PSS**) provide an excellent model of how Ca^{2+} regulation influences muscle metabolism and subsequent meat quality. Pigs with PSS are more prone to PSE development than normal pigs due to heightened susceptibility to preslaughter stress. When stressed, these pigs exhibit an exaggerated glycogenolysis, resulting in increased lactic acid accumulation, elevated body temperature, and muscle rigidity.

The well-established PSS condition (Vögeli et al., 1993) is manifested in a single autosomal recessive gene commonly referred to as the halothane (*Hal*) gene. The presence of the halothane gene in muscle results in a defective Ca^{2+} release channel associated with the sarcoplasmic reticulum (Mickelson et al., 1989). A point mutation in the 615th amino acid of the sarcoplasmic reticulum Ca^{2+} release channel protein (ryanodine receptor) is responsible for this defect (Fujii et al., 1991). In Hal^+ pigs, Ca^{2+} is released from the sarcoplasmic reticulum at twice the rate of release in normal pigs (Cheah and Cheah, 1976; Mickelson et al., 1989). Hence, increased cytosolic Ca^{2+} levels in the muscle cause an augmented rate of muscle metabolism and lactic acid accumulation. Many researchers have shown that the incidence of PSE is as much as fivefold higher in Hal^+ pigs than Hal^- pigs (Eikelenboom and Nanni-Costa, 1988; Lundström et al., 1989; Leach et al., 1996). These data suggest that PSE developed via the ryanodine receptor resides in the regulation of cellular Ca^{2+} concentrations early postmortem. More recently, Küchenmeister et al. (1999) showed that the Ca^{2+} sequestering ability of the sarcoplasmic reticulum from muscle of Hal^+ pigs is lower than that of Hal^- pigs. These data suggest that in addition to problems with Ca^{2+} gating in Hal^+ pigs, there is some uncertainty about the ability of the Ca^{2+} pump to operate in these pigs. It is not known to what extent the Ca^{2+} pump may be denatured from harsher pH and temperature conditions in Hal^+ pigs. Unfortunately, PSE may also develop in the absence of the *Hal* gene; hence, defective Ca^{2+} channels do not fully explain the PSE phenomenon. Consequently, data to determine the role of Ca^{2+} metabolism in the PSE development of Hal^- pigs are needed.

ATPase Activity. An "upstream" factor influencing rate of postmortem glycolysis is ATPase activity. Using parameters normally existing in postmortem muscle, Scopes (1974) developed a reconstituted system and ascertained that rate of glycolysis is directly proportional to the amount of ATPase activity. Those proteins responsible for utilizing ATP in muscle fibers are myofibrillar (myosin/actin) ATPase (mATPase), sarcoplasmic reticulum Ca^{2+}-ATPase (SR-ATPase), the plasmalemma Na^+,K^+-ATPase, plasmalemma Ca^{2+}-ATPase, and mitochondrial ATPase. Greaser et al. (1969) demonstrated that specific ATPase activity is similar in myofibrillar, mitochondrial, and heavy sarcoplasmic reticulum fractions immediately postmortem. Thus, given that myosin constitutes approximately 25% of all muscle proteins, the inference can be drawn that mATPase is likely the main factor regulating postmortem muscle ATPase activity, and that the other ATPases make only a minor contribution to overall rate of ATP degradation.

Because ATPase activity can regulate glycolysis by lowering cellular ATP concentrations and increasing ADP, it seems plausible to postulate that muscles developing PSE have higher mATPase activity. However, several studies have yielded data contradictory to this hypothesis. Greaser et

al. (1969) reported that mATPase activity increases postmortem in myofibril preparations from normal pig muscle, whereas mATPase activity decreases with time postmortem in myofibril preparations from PSE-generating pig muscle. Honikel and Kim (1985) later attempted to rule out the significance of mATPase by showing that sarcomeres of isolated myofibrils from PSE pork were unable to shorten, whereas those from normal pork maintained a functional contractile apparatus. Additionally, Honikel and Kim (1985) demonstrated that ATPase activity in the myofibrillar fraction in samples taken 1.5 h postmortem was lower in PSE than in normal muscle. Taken together, these data indicate that mATPase could not be driving postmortem glycolysis because myofibrils extracted from muscle with a rapid pH decline have reduced functionality.

Decreased functionality of PSE myofibrils is often perceived to occur from precipitation of sarcoplasmic proteins onto the surface of myofibrils (Bendall and Wismer-Pedersen, 1962). Yet, data from this study do not necessarily support this hypothesis. From Bendall and Wismer-Pedersen (1962) it can only be concluded that sarcoplasmic proteins denature, become insoluble, and subsequently appear in the myofibrillar fraction upon purification. This does not directly indicate that sarcoplasmic proteins precipitate onto myofibrils and reduce functionality in vivo.

At a glance, the results of Greaser et al. (1969) and Honikel and Kim (1985) are quite convincing regarding the role of mATPase in controlling glycolysis and form the basis for the argument against mATPase. Mechanisms triggering rapid glycolysis and PSE pork are thought to manifest within 1 h postmortem. Honikel and Kim (1985), however, did not sample muscle during this early time period. Therefore, the reduced ATPase activity and functionality of the PSE myofibrils were most likely the result of protein denaturation prior to sampling. Greaser et al. (1969) determined mATPase activity immediately postmortem, but they used pigs of unknown halothane gene status because a definitive test had not been developed. As described previously, the status of the *Hal* gene has major implications on the Ca^{2+} regulation and metabolic rate of muscle. Thus, such a potential confounding factor needs to be accounted for in investigating mechanisms by which glycolytic rate is enhanced and PSE develops.

Furthermore, assays from Greaser et al. (1969) were conducted at 25°C and 10^{-5} *M* Ca^{2+} concentrations, and levels were not titrated to simulate in vivo postmortem conditions. Because variations in Ca^{2+} concentration and temperature affect mATPase activity and myofibrillar functionality (Godt, 1974; Fabiato and Fabiato, 1978; Godt and Lindley, 1982), keeping these variables constant could have preferentially masked differences in ATPase activities between PSE and normal pork carcasses. Moreover, myofibrillar extractions were not indexed according to muscle fiber type (i.e., fast- vs slow-contracting myofibrils), which also alters mATPase activity (Cassens and Cooper, 1971). It is not known whether extraction protocols are selective for a specific subset of myofibrils. Indeed, subtle changes in contractile protein structure differ between fast- and slow-contracting muscle fibers and may have affected their results (Schiaffino and Reggiani, 1996). Therefore, definitive conclusions regarding the role of mATPase cannot be drawn from these studies.

Glycogenolytic Enzymes. Potential differences in key enzymes directly involved in the breakdown of muscle glycogen have been investigated as possible regulators of PSE development. The activities and characteristics of glycogen phosphorylase (**GP**), phosphofructokinase (**PFK**), and pyruvate kinase (**PK**) have been studied in an attempt to explain the accelerated rate of postmortem glycolysis in PSE muscle.

Glycogen phosphorylase catalyzes the breakdown of glycogen by sequentially removing glucose residues from the glycogen molecule. In skeletal muscle, GP exists in two forms: an active (phosphorylated) form (**GP *a***) and an inactive (unphosphorylated) form (**GP *b***). Early in vitro studies indicated a link between final pH and proportion of GP in the *a* form (Scopes, 1974). Thus, potential differences in total GP activity between PSE and normal muscle have been investigated in an attempt to explain the aberrant pH decline in PSE-prone muscle. Ensinger et al. (1982) reported that the activities of GP *a* and *b* were not different in muscles of normal and PSE animals, whereas Schwägele and Honikel (1988) demonstrated a fivefold higher activity of GP *a* and *b* in muscles prone to PSE development. Likewise, Ono et al. (1977) found an increased activity of GP *a* in PSE muscle but no difference in GP *b* activity. More recently, Schwägele et al. (1996b) found no significant differences between the kinetic or electrophoretic properties of GP from PSE and normal meat, and thus concluded that GP is not likely the primary regulator of the rapid postmortem metabolism in PSE-prone muscles.

Another enzyme worthy of attention is PFK, which irreversibly catalyzes the conversion of fructose 6-phosphate into fructose 1,6-bisphosphate and is considered the rate-limiting enzyme in the glycolytic pathway. In a reconstituted glycolytic system, PFK was found to still be active at pH 5.35 and 37°C, which are extreme PSE conditions (Scopes, 1974). Furthermore, Schwägele and Honikel (1988) demonstrated that total and specific PFK activity did not significantly change when sampled from longissimus muscles from pigs that ranged in 45-min pH from 5.3 to 6.8. Thus, the activation or inactivation of PFK is probably not the likely cause for the differing glycolytic rates observed in PSE and normal muscles, unless PFK from PSE muscle is more efficient at low pH than PFK from normal muscles. Such an increased activity at low pH has not been proven.

Pyruvate kinase (PK) irreversibly catalyzes the conversion of phosphoenol pyruvate to pyruvate. Pyruvate kinase was found to exhibit fourfold higher activity in PSE than in normal muscle, and optimal activity of PK extracted from PSE muscle is shifted toward lower pH values (Schwägele et al., 1996a). At pH 5.5, activity of PK from normal muscle is very low, whereas the PK from PSE muscles retains about 70% of its maximum activity. Based on isoelectric focusing on polyacrylamide gels, normal muscle has two isoforms of PK, whereas a third isoform is present in PSE muscle. The third isoform is highly phosphorylated and is most likely responsible for its increased activity, increased phosphoenol pyruvate utilization, and a shift in optimal activity in PSE

muscle (Schwägele et al., 1996a)**.** Existence of this isoform was not determined in living muscle, and it is not known whether this isoform is a causative factor or merely an artifact of the rapid glycolysis in PSE muscle. Thus, the role of PK in postmortem muscle has yet to be fully determined.

Overall, data thus far have failed to demonstrate that inherent differences in glycogenolytic enzymes explain why some pigs are more susceptible to a rapid rate of postmortem glycolysis and PSE development. Although it is possible to evaluate glycogenolytic enzyme activities and relate those values to ultimate meat quality, a more meaningful approach may be to determine the amount of glycolytic metabolites at discrete intervals postmortem. Such data would help describe the activities of glycolytic enzymes and allow tracking of metabolism in vivo that is related to meat quality.

Substrate Regulation. Although classic PSE development is the result of a rapid rate of glycolysis during the early (<1 h) postmortem period, PSE-like characteristics can also result from alterations in the extent of glycolysis. Extent of glycolysis, as determined by ultimate pH (**pH_u**), partially depends on muscle glycogen concentrations at death. Prolonged postmortem glycolysis results in the production of "acid meat" with low pH_u and is associated with high glycolytic potentials in muscle (Monin and Sellier, 1985). Glycolytic potential is a measure of glucose, glucose-6-phosphate, glycogen, and lactate in muscle and is commonly used as an indicator of the potential lactate formation of muscle tissue at time of slaughter (Monin et al., 1981). In particular, some Hampshire pigs possess high antemortem levels of muscle glycogen and their muscle undergoes extended postmortem glycolysis, resulting in a low pH_u and pale color (Sayre et al., 1963; Monin and Sellier, 1985; Monin et al., 1987). This is often termed the "Hampshire effect."

Elevated muscle glycogen levels are linked to a mutation in the *Rendement Napole* gene, which exists as two alleles: one dominant (RN^-) and one recessive (rn^+). Pigs with this mutation have extremely high glycolytic potentials (> 200 µmol/g tissue) and are more prone to develop PSE-like characteristics, such as a pale color and low water-holding capacity (LeRoy et al., 1990; Fernandez et al., 1992; Lundström et al., 1996). Specifically, white muscle fibers from RN^- carrier pigs may have up to 70% more glycogen in the sarcoplasmic and lysosomal compartments than do normal pigs (Estrade et al., 1993). Elevated muscle glycogen levels can be attributed to higher branching enzyme and glycogen synthase activities in the longissimus muscle of RN^- carrier pigs (Estrade et al., 1994). Regarding glycogen degradation, glycogen phosphorylase and debranching enzyme have similar activities in muscle biopsies from normal and RN^- pigs (Estrade et al., 1994). Surprisingly, Lebret et al. (1999) showed that pigs with the RN^-allele have a lower proportion of glycolytic muscle fibers and more oxidative fibers, thus implying a lower anaerobic glycolytic capacity. Because glycolytic fibers contain more glycogen than oxidative fibers (Fernandez et al., 1995), these results seem contradictory. Lebret et al. (1999) hypothesized that this discrepancy could be explained by a defect in glycogen metabolism in the white muscles of RN^- carriers. Data to explain this phenomenon need to be gathered.

High levels of glycogen alone do not fully explain the phenomenon of an extended glycolysis resulting in meat with low pH_u and PSE-like characteristics. Maribo et al. (1999b) and van Laack and Kauffman (1999) demonstrated that glycolytic potential levels only explain roughly 35 to 50% of the variation in pH_u. In theory, the duration and termination of postmortem glycolysis are controlled by substrate availability and enzyme activity. Thus, in order to understand better why muscles may undergo prolonged glycolysis postmortem, it is imperative to investigate factors regulating the cessation of glycolysis in systems in which glycogen is not limiting.

Termination of postmortem glycolysis is often thought to result from inactivation or denaturation of glycolytic enzymes due to low pH and high temperature conditions. Current data, however, do not define the precise enzyme or mechanism to support this hypothesis. Monin and Sellier (1985) demonstrated that pigs with high levels of muscle glycogen had normal rates of glycolysis, as indicated by normal pH values at 1 h postmortem. Thus, under normal carcass chilling conditions, pH and temperature profiles of muscle with elevated glycogen levels would be similar to those of normal muscle for an extended period postmortem until high glycogen muscle declines beyond the pH_u of normal muscle, by which time the carcasses are probably below denaturating temperature conditions. Unless inherent differences exist in the pH/temperature sensitivities of the glycolytic enzymes, muscle with high glycogen and normal muscle would hypothetically cease glycolysis at similar pH values. Yet data clearly show that meat from RN^- carriers has lower pH_u than meat from non- RN^- carriers (Monin and Sellier, 1985; Lundström et al., 1996; Enfält et al., 1997). Furthermore, Scopes (1974) suggested that at 37°C even the most pH-susceptible enzyme (PFK) is still active at pH 5.35 and that when glycogen is not limiting pH_u is related to glycogen phosphorylase and AMP deaminase activity. In order to determine more precisely mechanisms dictating a prolonged postmortem glycolysis and production of "acid meat," data regarding the inherent differences in the postmortem pH and temperature sensitivities of the various enzymes involved in postmortem metabolism from normal and RN^- muscle need to be generated.

Muscle Fiber Type. An intrinsic characteristic of muscle often overlooked in documenting regulating mechanisms responsible for PSE development is that of muscle fiber type. In general, "whiter" muscles such as the longissimus, gluteus medius, and biceps femoris, which have predominantly glycolytic fibers, are particularly susceptible to rapid postmortem glycolysis and PSE development (Briskey and Wismer-Pedersen, 1961; Merkel, 1971; Lundström et al., 1989; Warner et al., 1993). Conversely, "red" muscles, which have predominantly oxidative fibers, are much less susceptible to an accelerated postmortem glycolysis and PSE development (Briskey, 1964). Thus, muscle fiber type composition, at least drastic differences, plays a significant role in determining pork quality (reviewed by: Karlsson et al., 1999; Lefaucheur and Gerrard, 2000).

The link between muscle fiber type and PSE development stems from innate differences between the various fiber types with regard to ATPase activity, glycogen content, and glycolytic enzymatic capacity. Myosin ATPase activities vary across fiber types due to multiple isoforms of the myosin molecule, with white, glycolytic fibers possessing a higher ATPase activity than red, oxidative fibers. Thus, white fibers would be more prone to depleting ATP rapidly and triggering the accelerated glycolysis observed in PSE. Additionally, type IIB (glycolytic) fibers have greater concentrations of glycogen in resting muscle than more oxidative type I or IIA fibers (Karlsson et al., 1994; Fernandez et al., 1995) and are enzymatically better equipped to metabolize glycogen under anaerobic conditions (Cassens and Cooper, 1971; Essén-Gustavsson, 1983; Kiessling and Hansson, 1983). Distinguishing propensities of various fiber types to metabolize glycogen are evidenced by the fact that fasting and stress-induced glycogen depletion is both muscle type- and fiber type-dependent (Fernandez et al., 1994; Wittmann et al., 1994). Thus, susceptibility to PSE development in pigs may be partially due to differences in muscle metabolic and contractile properties as the result of muscle fiber type composition.

In addition to energy metabolism, Ca^{2+} metabolism is also fiber type-dependent. White, glycolytic muscle fibers possess a more extensive sarcoplasmic reticulum than red, oxidative fibers (Schiaffino et al., 1970; Peachey and Franzini-Armstrong, 1983). Consequently, release and sequestering of free Ca^{2+} probably varies with fiber type. This is vital because free Ca^{2+} levels within muscle fibers help mediate and regulate overall cellular ATPase activity and glycolysis. Thus, muscle fiber type can be linked to postmortem muscle pH decline through elevated intracellular Ca^{2+} concentrations that are triggered during the slaughter process. Studies to delineate these mechanisms in postmortem muscle are necessary to understand fully how variations in muscle fiber type profiles alter postmortem metabolism and influence PSE development.

Models

In order to investigate mechanisms responsible for PSE development, it is beneficial to have established models that can routinely generate PSE pork. Methodologies to induce the protein denaturation and subsequent PSE quality attributes may potentially be useful to delineate underlying mechanisms responsible for the aberrant postmortem metabolism in PSE carcasses. Two such methods have been investigated: retarding temperature decline (**RTD**) and electrical stimulation (**ES**).

The earliest success in artificially inducing PSE was achieved by holding muscle at 37°C (Bendall and Wismer-Pedersen, 1962; Bodwell et al., 1966). Bendall and Wismer-Pedersen (1962) demonstrated that low water retention and denaturation of sarcoplasmic proteins characteristic of PSE muscle can be induced in normal muscle by allowing it to pass into rigor at 37°C. Similarly, McCaw et al. (1997) showed that incubation of normal porcine muscle at high temperatures (40°C) for 4 to 8 h increased pH decline and resulted in a paler color and a reduced water-holding capacity.

Theoretically, ES generates PSE-like characteristics in muscle by accelerating postmortem glycolysis and hastening pH decline. Indeed, early studies demonstrated that ES hastens the rate of postmortem pH decline in muscle samples normally exhibiting a slow rate of pH decline (Hallund and Bendall, 1965; Forrest and Briskey, 1967). In whole carcass models, ES data have been inconsistent. Bowker et al. (1999) found that PSE-like characteristics can be induced in muscle by electrically stimulating intact pork carcasses early postmortem (3 min). In this study, ES induced pH declines similar to those reported for naturally occurring PSE. Similarly, Maribo et al. (1999a) demonstrated that ES results in a faster pH decline, increased drip loss and reflectance value, and an overall increase in the incidence of PSE. Contrarily, others have shown that postmortem ES of pork carcasses accelerates pH decline but has minimal effect on pork quality traits (Westervelt and Stouffer, 1978; Johnson et al., 1982; Gigiel and James, 1984; Taylor et al., 1995). Discrepancies in the data can be partially explained by accounting for differences in level of ES and postmortem time of ES. With this in mind, data indicate that in whole carcass models the earlier postmortem and the more severe the level of ES, the more drastic the effect on rate of pH decline and meat quality attributes.

Nonetheless, use of RTD and ES models to investigate mechanisms of PSE development has limitations. Ideally, models to study such mechanisms would induce, in normal musculature, pH and temperature profiles similar to those observed in naturally occurring PSE muscle during the critical early (<1 h) postmortem period. Although RTD models effectively mimic the elevated temperature of PSE development, RTD models fail to accelerate the rate of glycolysis in normal muscle to that observed in early postmortem PSE muscle (McCaw et al., 1997). Although high-voltage ES early postmortem can effectively simulate pH and temperature declines of PSE within the 1st h postmortem (Bowker et al., 1999), other factors associated with carcass stimulation may confound data from ES models. Electrical stimulation of carcasses causes muscular contraction not necessarily evident in naturally occurring PSE. Such contractions may cause physical disruption of cell membranes and loss of sarcomere integrity that subsequently affect various pork quality attributes. In beef muscle ES has been demonstrated to cause a considerable degree of physical disruption of myofibrils (Savell et al., 1978; McKeith et al., 1980; Will et al., 1980), but such data are lacking on whole pork carcass models. Furthermore, the degree to which different levels of ES cause an aberrant efflux of Ca^{2+} from the sarcoplasmic reticulum and accelerate glycolysis in whole carcass models has not been determined. Such data could help illuminate the mechanism by which ES affects pH decline and help explain some of the discrepancies in the ES data. Additionally, data are needed to demonstrate that protein denaturation patterns generated by RTD and ES models effectively simulates the pattern observed in classic PSE pork (Warner et al., 1997).

Implications

The pale, soft, and exudative (PSE) condition typically results from an accelerated rate of postmortem glycolysis. Understanding precise regulators of aberrant postmortem metabolism, however, requires data documenting cause and effect mechanisms rather than merely correlating variables pertaining to PSE development. The retarding temperature decline and electrical stimulation models are potential tools to aid researchers in identifying mechanistic factors contributing to PSE development. The ATPase activity, glycolytic enzyme activity, Ca^{2+} regulation, and muscle glycogen content affect rate and extent of postmortem glycolysis. Differences in such factors individually fail to explain fully the aberrant metabolism leading to PSE. One approach that integrates these factors is to investigate effects of muscle fiber type on postmortem mechanisms of PSE development. By generating data to determine basic biochemical mechanisms rendering muscle susceptible to PSE development, researchers can better determine methods to reduce PSE.

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Notes

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